

NOTES

HYPERTENSION & HYPOTENSION

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

- Changes of blood pressure above (hyper-) or below (hypo-) normal (120/80mmHg)

CAUSES

- Mostly idiopathic, but can include various causes: impaired regulatory pathways (hormonal/neurologic disorders), heart disease, kidney disease, medications etc.

SIGNS & SYMPTOMS

- Can be asymptomatic/include symptoms according to underlying cause, degree of change in blood pressure
 - Hypertension:** range from headache, dyspnea, to blurred vision, oliguria, seizures
 - Hypotension:** range from fatigue, pallor to syncope

DIAGNOSIS

OTHER DIAGNOSTICS

- Blood pressure cuff (sphygmomanometer) or arterial catheter

TREATMENT

- Hypotension:** generally requires no treatment
- Hypertension:** treated according to degree, treat underlying cause if present

MEDICATIONS

- Hypertension:** beta blockers, diuretics, ACE inhibitors, calcium channel blockers, etc.

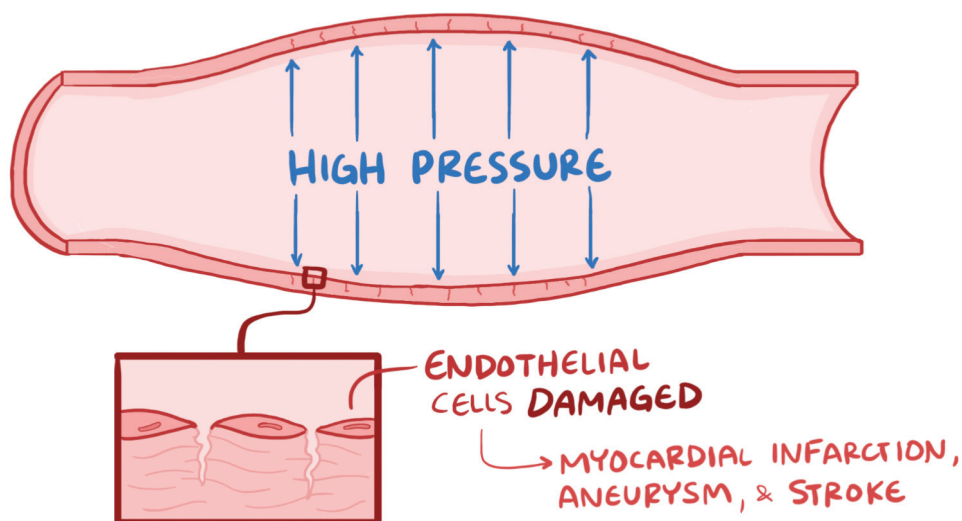


Figure 11.1 Illustration depicting endothelial damage caused by hypertension.

VISIT...

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Caliente.COM

HYPERTENSION

osms.it/hypertension

PATHOLOGY & CAUSES

- Condition in which blood pressure is regulated maladaptively, elevating blood pressure over 140/90mmHg
- **Isolated systolic hypertension:** systolic blood pressure is elevated, diastolic is not
- **Isolated diastolic hypertension:** diastolic blood pressure is elevated, systolic is not

CAUSES

Primary/essential hypertension

- Most cases (90%)

Secondary hypertension

- Known etiology, often reversible
- **Renovascular hypertension:** anything partially obstructing blood flow to kidneys (e.g. atherosclerosis, vasculitis, fibromuscular dysplasia).
 - Decreased blood flow to kidneys → kidneys secrete renin → renin converts angiotensinogen to angiotensin I → angiotensin converting enzyme converts angiotensin I to angiotensin II (active) → angiotensin II effects:
 - **Vasoconstriction:** directly increases blood pressure
 - **Stimulation of sodium reabsorption:** increases water reabsorption
 - Stimulation of adrenal cortex to release aldosterone from adrenal cortex → aldosterone increases reabsorption of sodium + water → increased volume → high blood pressure
- **Primary hyperaldosteronism:** increased aldosterone → increased reabsorption of sodium + water → increased volume → increased blood pressure
- **Nonsteroidal anti-inflammatory drugs (NSAIDs)** → inhibit cyclooxygenase in kidneys → ↓ production of PGE-2 (renal vasodilator) → vasoconstriction of afferent arterioles in kidneys → ↓ renal blood flow,

glomerular filtration rate → ↓ secretion of sodium + water → ↑ volume → ↑ blood pressure

- **Preeclampsia/eclampsia in pregnancy:** mechanism unknown
- **Coarctation of the aorta:** low pressure past coarctation → low renal perfusion → activation of renin angiotensin-aldosterone system (RAAS) → secondary hypertension
- **Cushing's syndrome:** combination of several pathophysiological mechanisms (e.g. elevated cortisol) that regulate plasma volume, cardiac output, peripheral vascular resistance
- **Chronic kidney disease:** fluid overload, sodium retention



MNEMONIC: RHNECCK

Causes of Secondary hypertension

Renovascular hypertension
1° Hyperaldosteronism
NSAIDs
Pre-**E**clampsia / **E**clampsia
Coarctation of the aorta
Cushing's syndrome
Kidney Disease (chronic)

RISK FACTORS

Primary hypertension

- Risk increases with age, biological male, obesity, stress, sedentary lifestyle, family history of hypertension
- **Race (in decreasing order of risk):** African descent > white European descent > Asian descent
- **Diet:** excessive sodium, alcohol intake
- Abnormal lipid panel (high low-density lipoproteins, low high-density lipoproteins, high triglycerides)

Secondary hypertension

- Atherosclerosis: Elderly biological males
 - Atherosclerosis → renal stenosis → less blood flow to renal arteries → activation of RAAS → renovascular hypertension

COMPLICATIONS

- Increased risk of atherosclerosis, arteriosclerosis
- Arteriolar rarefaction: loss of arterioles
- Coronary artery disease, left ventricular hypertrophy, atrial fibrillation, stroke, hypertensive nephropathy, retinopathy, aortic dissection, aneurysms

SIGNS & SYMPTOMS

- Vast majority of cases asymptomatic
- May experience headache, dyspnea
- Renal bruit in secondary hypertension due to renal artery stenosis
- Hypertensive retinopathy

DIAGNOSIS**OTHER DIAGNOSTICS**

- Non-invasive/invasive blood pressure monitoring
 - High blood pressure: at least 2 separate measurements with blood pressure > 140/90mmHg

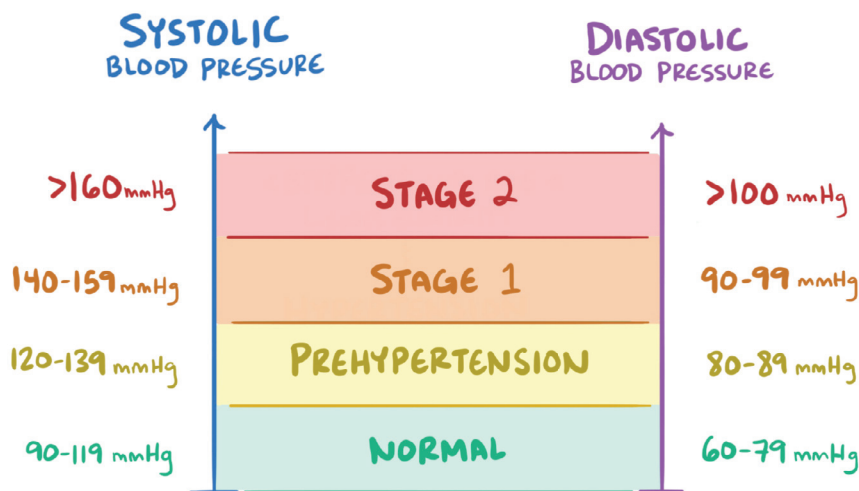


Figure 11.2 Illustration depicting stages of hypertension.

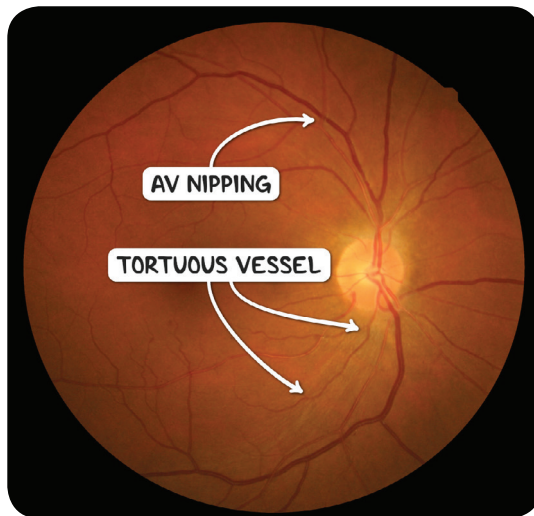


Figure 11.3 Retinal photograph demonstrating changes of hypertensive retinopathy (AV nipping and tortuous vessels).

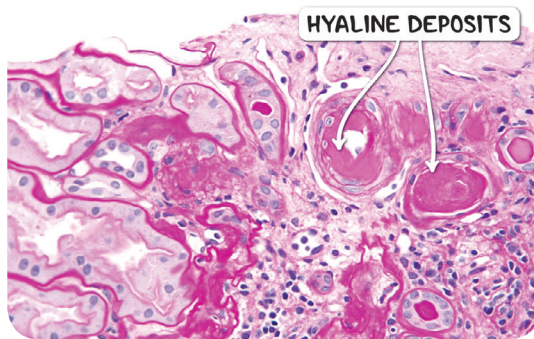


Figure 11.4 Histological appearance of renal artery hyaline arteriosclerosis; a manifestation of hypertensive renal disease.

TREATMENT

- Reduce BP <140/90mmHg with lifestyle modification first, then medical treatment

MEDICATIONS

Monotherapy/together

- **Thiazide-type diuretics:** reduce blood volume by increasing excretion of sodium, water
- **Angiotensin-converting enzyme (ACE) inhibitors:** block ACE from converting angiotensin I to angiotensin II → blocks RAAS → dilates arteries, decreases blood volume
 - Lower levels of angiotensin II → vasodilation
- **Dihydropyridine calcium channel blockers:** disrupt movement of calcium through calcium channels in blood vessel walls → vasodilation

Other agents

- **Angiotensin II receptor blockers (ARBs):** prevents the vasoconstrictive effects of angiotensin II by blocking its receptors → lowering blood pressure
 - Used when individuals get a cough from ACE inhibitors
- **Beta receptor blockers:** decrease contractility, heart rate
- **Alpha-2 agonist:** stimulates alpha-2 receptors → decreases sympathetic activity → decreased blood pressure, heart rate
- **Renin inhibitor:** aliskiren
 - Lower levels of angiotensin I
- **Hydralazine:** elicits direct vasodilation of vascular smooth muscle, useful in pregnancy

OTHER INTERVENTIONS

- Low sodium diet, exercise, quit smoking, limit alcohol, maintain healthy weight

HYPERTENSIVE EMERGENCY

osms.it/hypertensive-emergency

PATHOLOGY & CAUSES

- Elevated blood pressure > 180/120mmHg with signs of acute end organ damage (e.g. encephalopathy, stroke, papilledema, myocardial infarction, heart failure, microangiopathic hemolytic anemia, etc.)
- Complication of poorly managed hypertension

RISK FACTORS

- Kidney failure, renovascular hypertension, stimulant abuse, medication non-adherence
- More common in young adults, particularly those of African descent

COMPLICATIONS

- Neurological complications (stroke, seizures), myocardial infarction, kidney failure, permanent blindness, pulmonary edema

SIGNS & SYMPTOMS

- Blood pressure > 180/120mmHg, signs of end-organ damage
 - Blurred vision, altered mental state, chest pain, headache, nausea, vomiting, numbness in extremities, oliguria, seizure, dyspnea, weakness, papilledema

DIAGNOSIS

OTHER DIAGNOSTICS

- Sphygmomanometer
 - Blood pressure > 180/120mmHg
- Evaluation to identify at-risk target organ
 - Electrocardiography (heart)
 - Chest X-ray (heart, lungs)
 - Urinalysis (kidneys)
 - Serum electrolytes, serum creatinine (kidneys)
 - Cardiac enzymes (heart)
 - CT scan of brain (if brain suspected → neurologic symptoms, retinopathy)
 - Contrast-enhanced CT scan of chest (if aortic dissection suspected)

TREATMENT

- Treatment varies case-by-case, dependent on affected organ

MEDICATIONS

- Unwise to lower blood pressure too quickly/ too much, as this can lower cerebral perfusion excessively
 - Most cases: mean arterial pressure (MAP) should be reduced using intravenous medication 10–20 % in first hour, then 5–15% over the following 23 hrs. Specific medications used dependent on case

OTHER INTERVENTIONS

- Exceptions to most cases: acute phase ischemic stroke (not lowered unless specific conditions met)
 - Acute aortic dissection (rapid lowering), intracerebral hypertension (variable)

HYPOTENSION

osms.it/hypotension

PATHOLOGY & CAUSES

- Condition in which arterial blood pressure drops below 90/60mmHg
- Physiological in some cases (professional athletes); considered pathologic if symptomatic
- Not a distinctive disease, but a manifestation of various conditions
- **Orthostatic hypotension:** hypotension caused by standing up from a sitting/lying position

CAUSES

Hypovolemia

- Fluid loss (hemorrhage, diarrhea, vomiting), low fluid intake (starvation, oligodipsia), endocrine disorders, anemia

Heart disease

- Cardiomyopathies, heart valve disease (e.g. mitral stenosis), congestive heart failure, myocardial infarction, arrhythmias

Medications

- Most commonly cause orthostatic hypotension
- Excessive use of diuretics, alpha/beta blockers, nitrate preparations, calcium channel blockers, angiotensin II (AT1) receptor blockers, antidepressants

Neurological disorders

- Spinal cord injury resulting in ↓ sympathetic output or ↑ parasympathetic output
- Dysautonomia (intrinsic autonomic system dysfunction), Parkinson's disease

COMPLICATIONS

- Ischemia
- If severe, can lead to shock

SIGNS & SYMPTOMS

- Lightheadedness, fatigue, pallor, confusion
- Significant hypotension → syncope

DIAGNOSIS

OTHER DIAGNOSTICS

- Evaluation of blood pressure with sphygmomanometer/arterial catheter
 - Systolic blood pressure < 90mmHg
 - Diastolic blood pressure < 60mmHg
 - Mean arterial pressure < 65mmHg
- Orthostatic hypotension
 - Drop in 20mmHg of systolic pressure/10mmHg of diastolic pressure when standing up from a sitting/lying position

TREATMENT

- Asymptomatic hypotension does not require treatment
- Treat underlying cause